

Supplementary Table 1. Details regarding the cell lines used in this study.

Cell line ID	Age (yrs)	Sex	Institution	Molecular status	Tests	Date of tissue collection	Culture medium
SU-DIPG IV	3	F	Stanford University	DMG, H3.1 K27M	Sanger, STR, WB	Received 2016	TSM
SU-DIPG XIII	6	F	Stanford University	DMG, H3.3 K27M	Sanger, STR, WB	Received 2016	TSM
SU-DIPG XVII	8	M	Stanford University	DMG, H3.3 K27M	Sanger, STR, WB	Received 2016	MHM
SF8628	3	F	Millipore	DMG, H3.3 K27M	Sanger, STR, WB	Purchased 2019	dBT
NHA	–	–	Lonza	–	–	Purchased 2019	ABM

Abbreviations: DMG = diffuse midline glioma, STR = short tandem repeat, WB = western blot.

Supplementary Table 2. Media composition for culture of patient-derived cell lines.

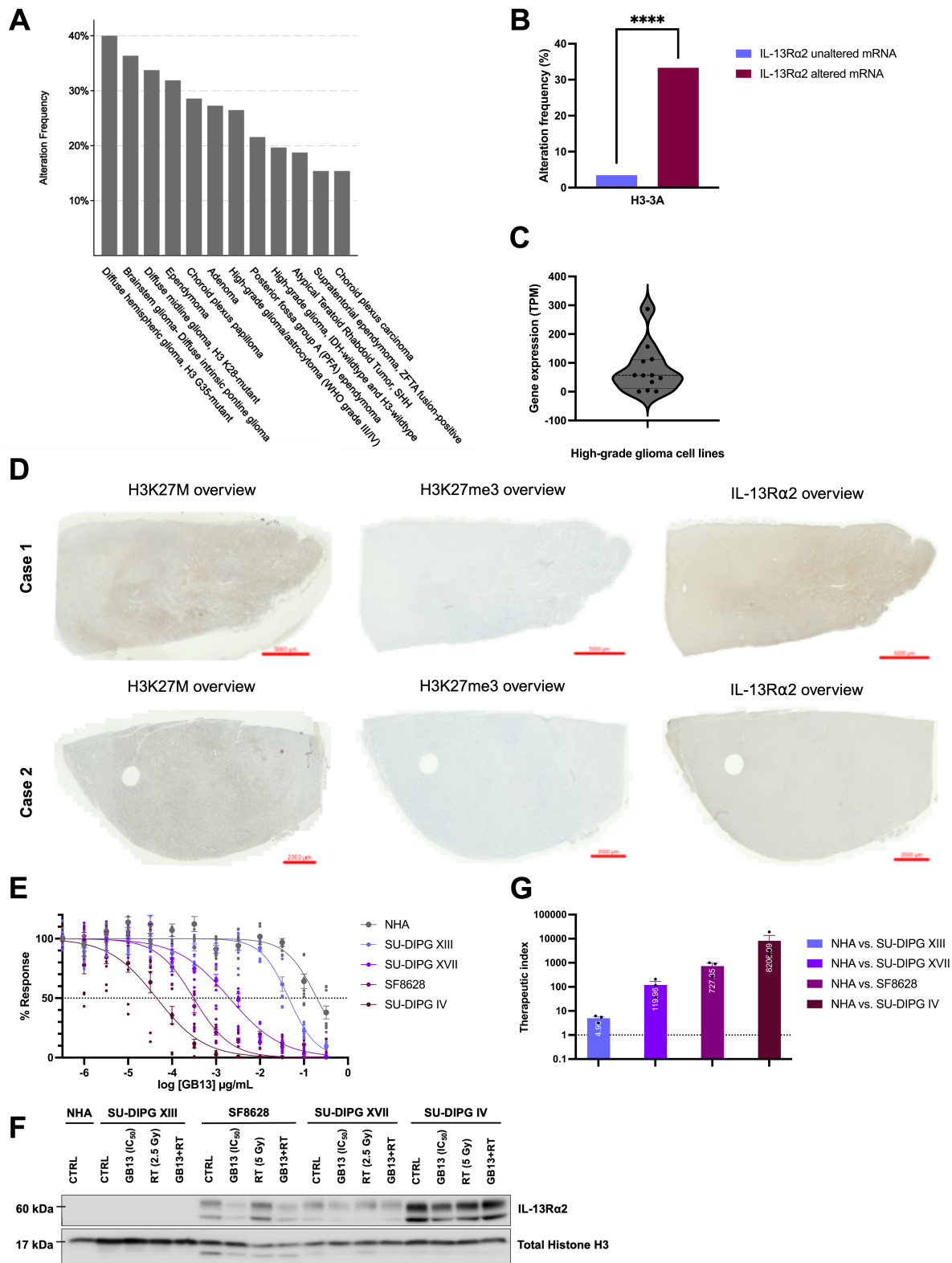
Cell Lines	Media name	Media contents
SU-DIPG IV, SU-DIPG XIII	Tumor stem cell medium (TSM)	Neurobasal(-A) (Life Technologies), DMEM/F12 (Life Technologies), B27(-A) (Life Technologies), 4 µg/mL heparin (Sigma Aldrich), 20 ng/mL human EGF (PeproTech US), 20 ng/mL human b-FGF (PeproTech US), 20 ng/mL human PDGF AA with 20 ng/mL human PDGF BB (Shenandoah Biotechnology), and 1% penicillin/streptomycin (Life Technologies).
SU-DIPG XVII	Serum-free complete medium (MHM)	DMEM/F12 (HyClone), 25 mM glucose (Sigma-Aldrich), 8.9 mM sodium bicarbonate (Life Technologies), 2 mM glutamine (Life Technologies), 4 mM HEPES (Life Technologies), 1% penicillin/streptomycin (Life Technologies), N2 supplement (Life Technologies), 4 µg/mL heparin (Sigma Aldrich), 20 ng/mL human EGF (PeproTech US), 20 ng/mL human b-FGF (PeproTech US), and 20 ng/mL human PDGF AA with 20 ng/mL human PDGF BB (Shenandoah Biotechnology).
SF8628	Delayed brain tumor (dBT) medium	DMEM/F12 (HyClone), 10% FBS, and 1% penicillin/streptomycin (Life Technologies).
NHA	Astrocyte growth medium (AGM)	ABM Basal Medium (CC-3187; Lonza), AGM SingleQuots Supplement Pack (CC-4123; Lonza) containing: 15 mL FBS, 5 mL L-glutamine, 0.5 mL GA-1000, 0.5 mL ascorbic acid, 0.5 mL HEGF, and 1.25 mL insulin.

Supplementary Table 3. Antibodies used for western blot, immunocytochemistry/immunofluorescence, and immunohistochemistry.

Antibodies	Source	Identifier	Dilution for WB	Dilution for ICC/IF	Dilution for IHC
α -Tubulin	Sigma-Aldrich	T9026	1:20000		
BAK	Sigma-Aldrich	06-536	1:1000		
CD68	Abcam	Ab125212			1:800
Cleaved Caspase 3	Cell Signaling	CS-9661			1:200
Cleaved Caspase 3	Cell Signaling	CS-9664S	1:1000		
C-Myc	Cell Signaling	CS-18583	1:1000		
Cleaved PARP	Cell Signaling	CS-5625S	1:500		
GAPDH	Cell Signaling	CS-2118S	1:3000		
H3K27M	Abcam	Ab190631	1:3000		1:500
H3K27me3	Cell Signaling	CS-9733S	1:3000		1:200
Total Histone H3	Cell Signaling	CS-3638S	1:3000		
IL-13R α 1	Abcam	Ab79277	1:1000		
IL-13R α 2	ABclonal	A2043			1:100
IL-13R α 2	Cell Signaling	CS-85677	1:1000		
IL-13R α 2	Santa Cruz	sc-134363			
Ki-67	Invitrogen	I-14-5698-82			1:500
NeuN	Sigma-Aldrich	MAB377			1:600
Mcl-1	Rockland	600-401-394	1:1000		

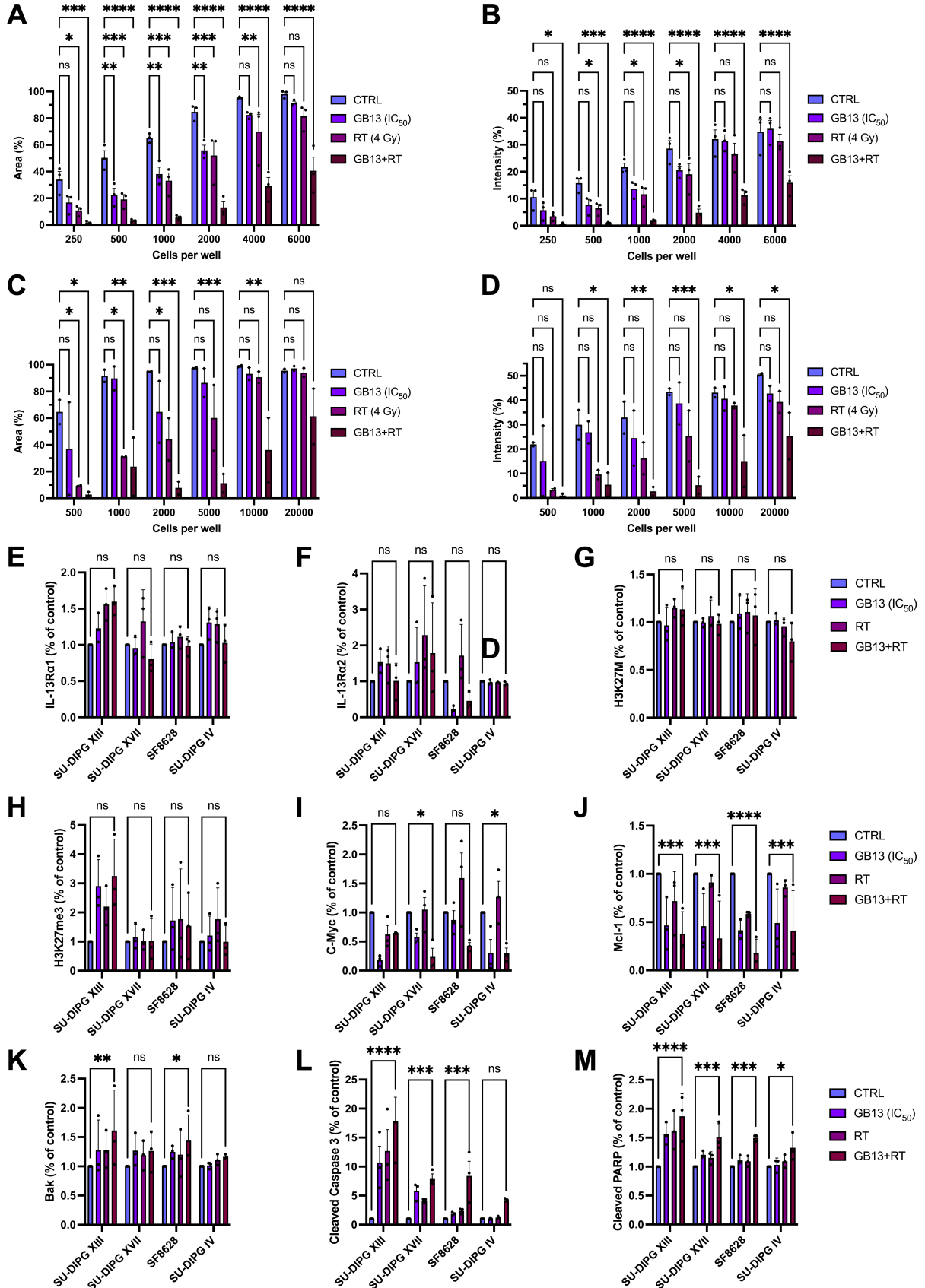
γH2AX	Cell Signaling	CS-80312	1:1000		
γH2AX	Cell Signaling	CS-2577		1:300	
Total H2AX	Cell Signaling	CS-2595	1:1000		

Abbreviations: WB = western blot, ICC = immunocytochemistry, IF = immunofluorescence, IHC = immunohistochemistry.

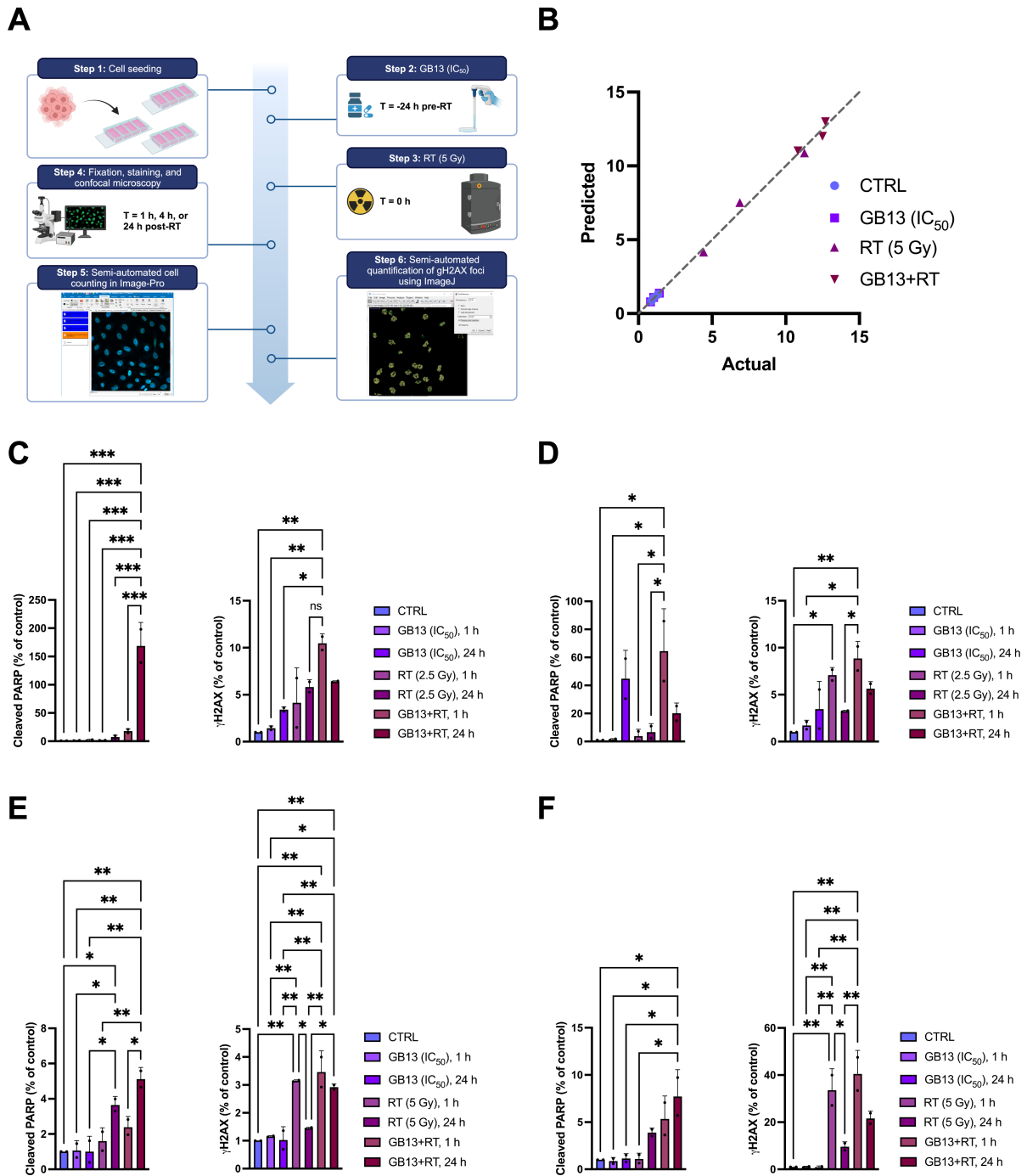


Supplementary Figure 1. Exploration of multidimensional patient cancer genomics data sets, DIPG patient tissue samples, and pharmacological interrogation of patient-derived cell lines identifies interleukin 13 receptor subunit alpha 2 (IL-13Ra2) as a

prevalent and targetable alteration for pseudomonas exotoxin A (PE)-based immunotoxin therapy in multiple pediatric brain cancers, including diffuse intrinsic pontine glioma (DIPG). **(A)** Genomic analysis of patient tissue showing the frequency of IL-13R α 2 alterations, including structural variant data, mutation data, copy number alterations (CNA) data, and mRNA data, in different cancer types. **(B)** Malignant glioma diagnoses and the percentage of *H3-3A* mutation co-occurrence based on IL-13R α 2 alteration or un-alteration. *H3-3A* is one of the highest co-occurring perturbations with IL-13R α 2. When IL-13R α 2 is altered, the *H3-3A* mutation occurs 33.3% of the time, compared to only 3.5% of the time in IL-13R α 2 unaltered samples. Statistical significance denoted as ****, $P \leq 0.0001$; analyzed via unpaired t test. Taken from the cBio Cancer Genomics Portal (<http://cbioportal.org>) and the Open Pediatric Brain Tumor Atlas (OpenPBTA) dataset in PedcBioPortal (<https://pedcbioportal.org>)^{1,3-5}. **(C)** IL-13R α 2 gene expression pattern in a large panel of patient-derived high-grade glioma cell lines, including DIPG. **(D)** Immunohistochemistry (IHC) images of autopsy-derived formalin-fixed tumor specimens from DIPG patients (H3K27M, H3K27me3, and IL-13R α 2). Scale bars: 1 $\times$: 5000 and 2000 μ m for Case 1 and 2, respectively. **(E)** GB13 (IL13.E13K-PE4E) cell viability analysis in normal human astrocytes (NHA) versus patient-derived DIPG cells using a 12-point dose-response experimental setup. Results are the mean $\pm$ SEM of 3 independent experiments. **(F)** Western blot of normal human astrocyte (NHA) and DIPG cell lines with increasing IL-13R α 2 expression (SU-DIPG XIII, SU-DIPG XVII, SF8628, and SU-DIPG IV) treated for 72 hours with vehicle control, GB13 alone at IC₅₀, RT alone, or RT plus GB13 at IC₅₀ (added 24 hours pre-RT). Total Histone H3 is the loading control. **(G)** Therapeutic indices of GB13, a measure of the ratio of the dose that produces toxicity to the dose that produces a clinically desired or effective response, were calculated based on the IC₅₀ in NHA over the IC₅₀ of individual DIPG cell lines.

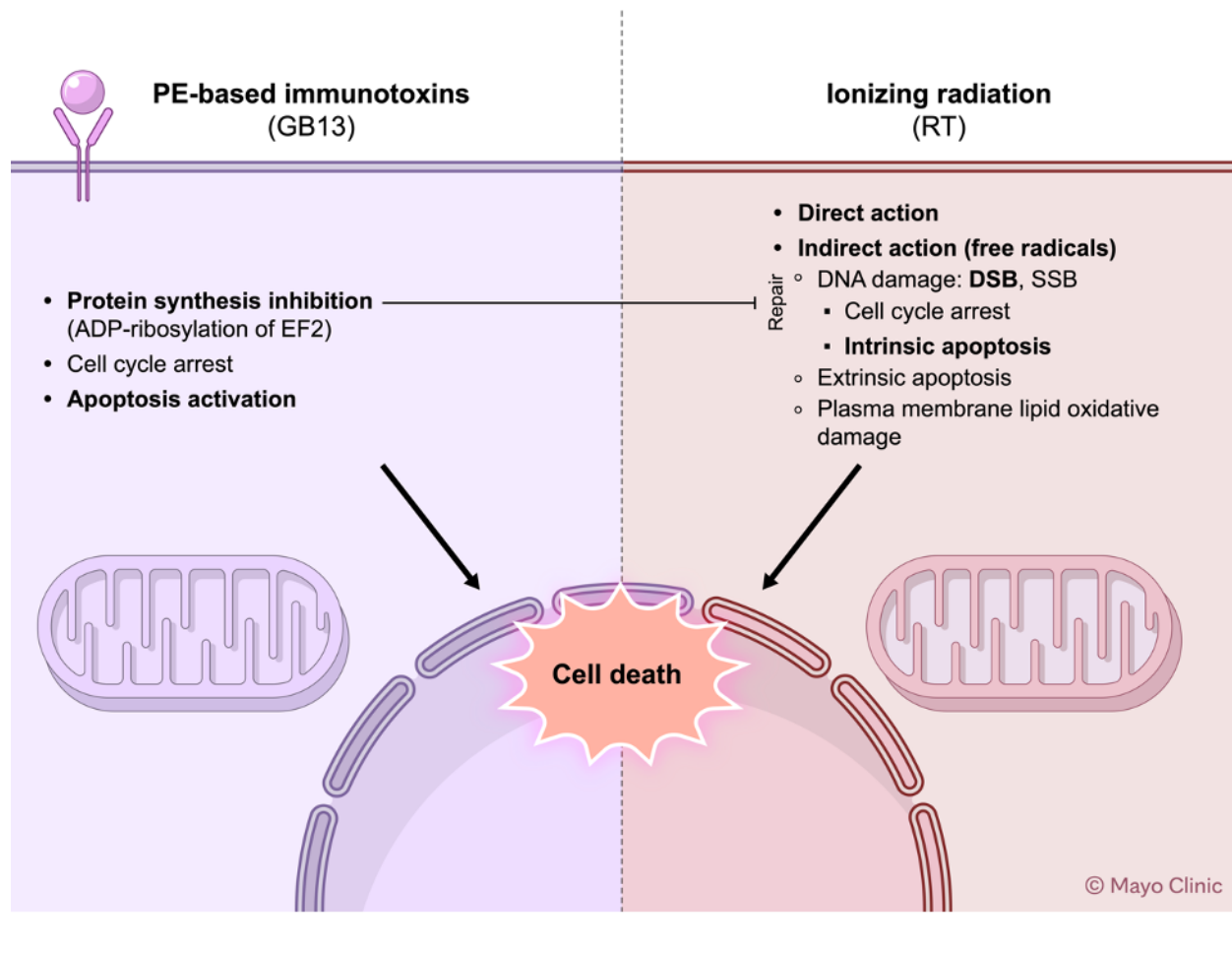


Supplementary Figure 2. Concurrent GB13 plus radiotherapy (RT) potentiates the *in vitro* efficacy and on-target effects of either treatment alone in cultured DIPG cells. Clonogenic survival for **(A-B)** 250-6000 SF8628 cells/well and **(C-D)** 500-20000 SU-DIPG IV cells/well in a 6-well plate treated with vehicle control, GB13 alone at IC₅₀, 4 Gy of RT alone, or GB13 at IC₅₀ 24 hours pre-RT plus 48 hours post-RT (4 Gy), rinsed with PBS, media changed, and incubated for colony formation. Image data were analyzed with ColonyArea using the **(A, C)** colony area percentage and **(B, D)** colony intensity percentage tools⁶. Results are the mean ± SEM of 3 independent experiments. ns: P ≥ 0.05; *: P ≤ 0.05; **: P ≤ 0.01; ***: P ≤ 0.001; ****: P ≤ 0.0001; two-way ANOVA. **(E-M)** Western blot quantification of DIPG cell lines with increasing IL-13Rα2 expression (SU-DIPG XIII, SU-DIPG XVII, SF8628, and SU-DIPG IV) treated with vehicle control, GB13 alone at IC₅₀, RT alone, or RT plus GB13 at IC₅₀. Cells were exposed to GB13 or vehicle for a total of 72 hours, with a single irradiation dose administered 24 hours after drug treatment initiation. Treatment-dependent protein level changes of **(E)** IL-13Rα1, **(F)** IL-13Rα2, **(G)** H3K27M, **(H)** H3K27me3, **(I)** C-Myc, **(J)** Mcl-1, **(K)** Bak, **(L)** cleaved caspase 3, and **(M)** cleaved PARP-1 were normalized to vehicle control. Results are the mean ± SD of 3 independent experiments. ns: P ≥ 0.05; *: P ≤ 0.05; **: P ≤ 0.01; ***: P ≤ 0.001; ****: P ≤ 0.0001; two-way ANOVA.

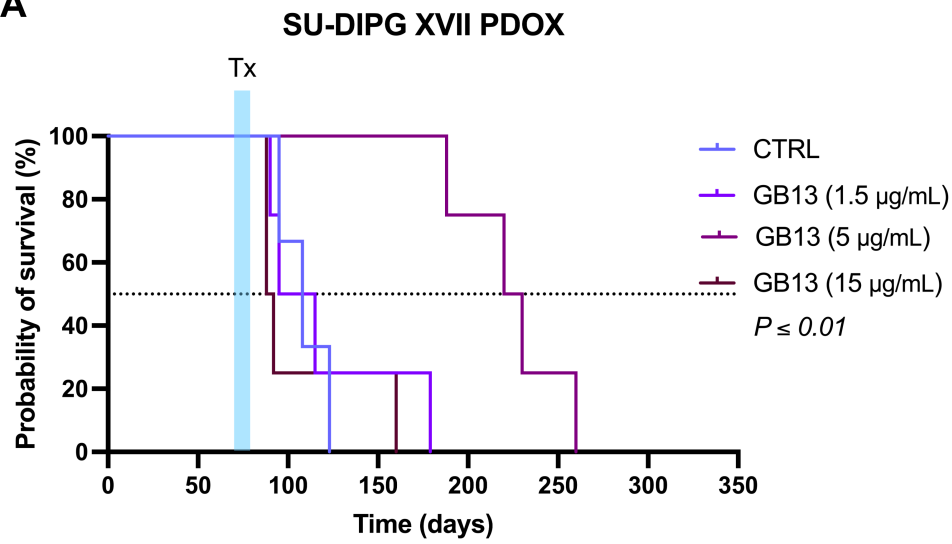
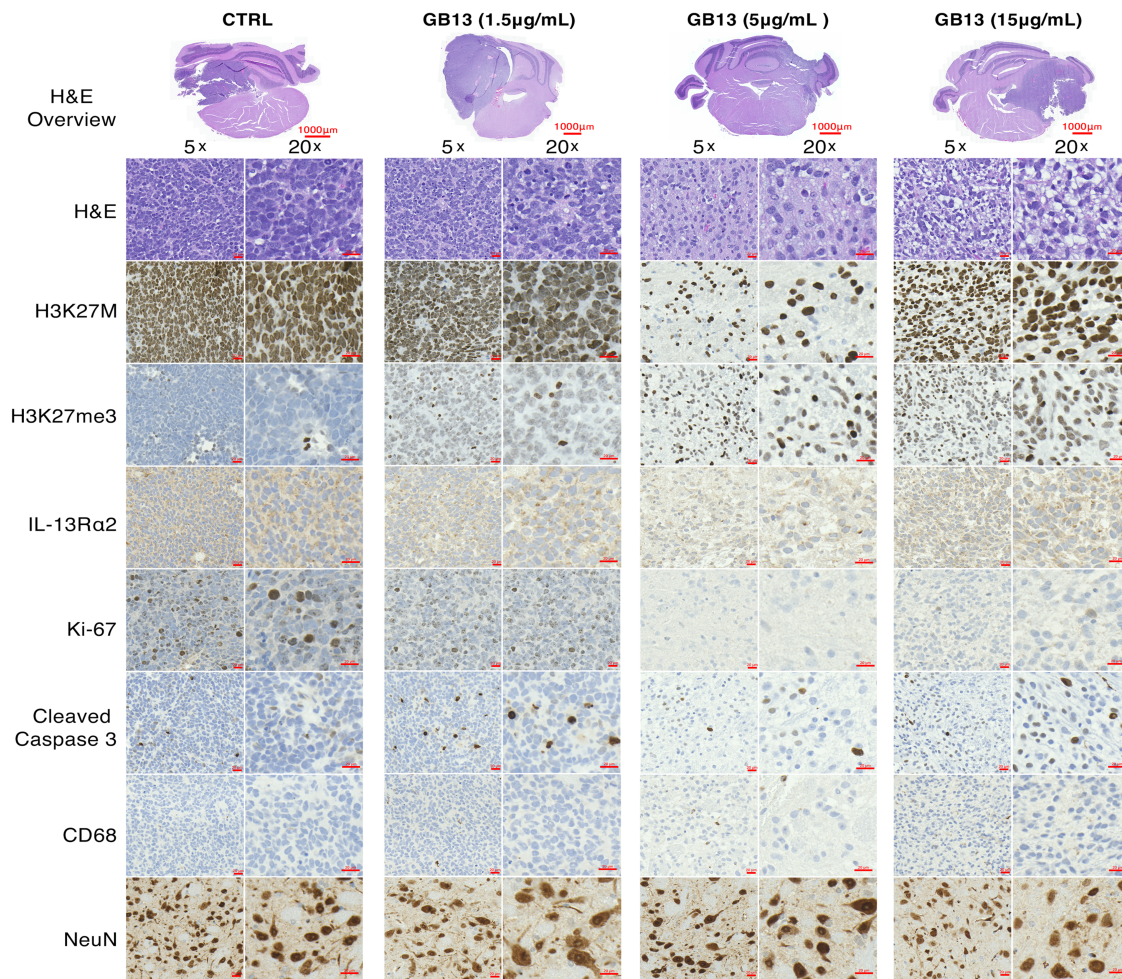


Supplementary Figure 3. GB13 increases RT-induced DNA damage and DIPG cell killing. **(A)** Schema for experimental workflow of immunofluorescence-based, semi-automated γ H2AX foci quantification in SF8628 cells treated with vehicle control, GB13 alone at IC_{50} , 5 Gy of RT alone, or 5 Gy RT plus GB13 at IC_{50} (added 24 hours pre-RT). Cells were fixed and processed 1 hour, 4 hours, or 24 hours after RT or GB13 monotherapy. **(B)** QQ plot of Shapiro–Wilk normality test demonstrates Gaussian distribution of γ H2AX foci data (vehicle control: $W = 0.96$; GB13: $W = 0.97$; RT: $W = 0.97$; GB13+RT: $W = 0.83$). **(C–F)** Western blot quantification of the time- and treatment-

dependent implications of GB13 alone, RT alone, or RT plus GB13 at IC₅₀ (added 24 hours pre-RT) on DNA damage repair and apoptosis induction in **(C)** SU-DIPG XIII, **(D)** SU-DIPG XVII, **(E)** SF8628, and **(F)** SU-DIPG IV cells. Samples were collected 1 hour and 24 hours after treatment start and normalized to vehicle control. Results are mean $\pm$ SD of 3 independent experiments. *: $P \leq 0.05$; **: $P \leq 0.01$; ***: $P \leq 0.001$; one-way ANOVA.

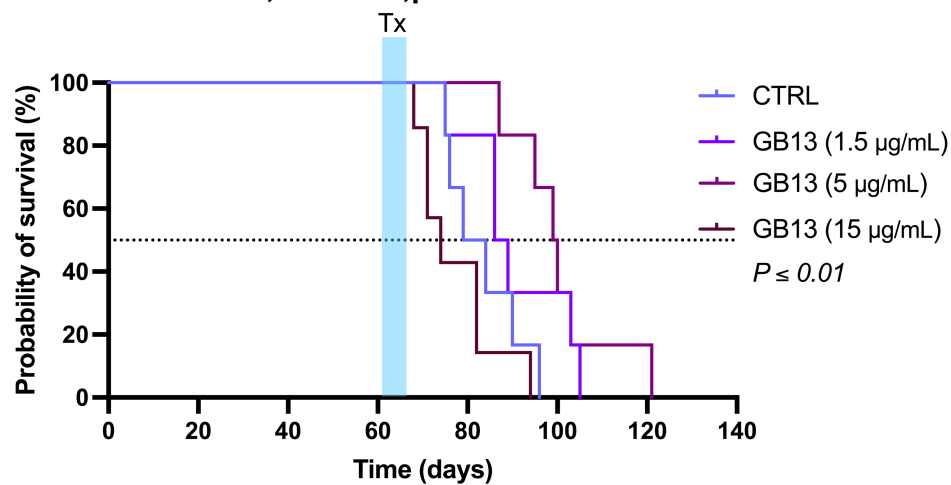


Supplementary Figure 4. Overview of the proposed mode of action for concurrent ionizing radiation (e.g., RT) and PE-based immunotoxins (e.g., GB13).

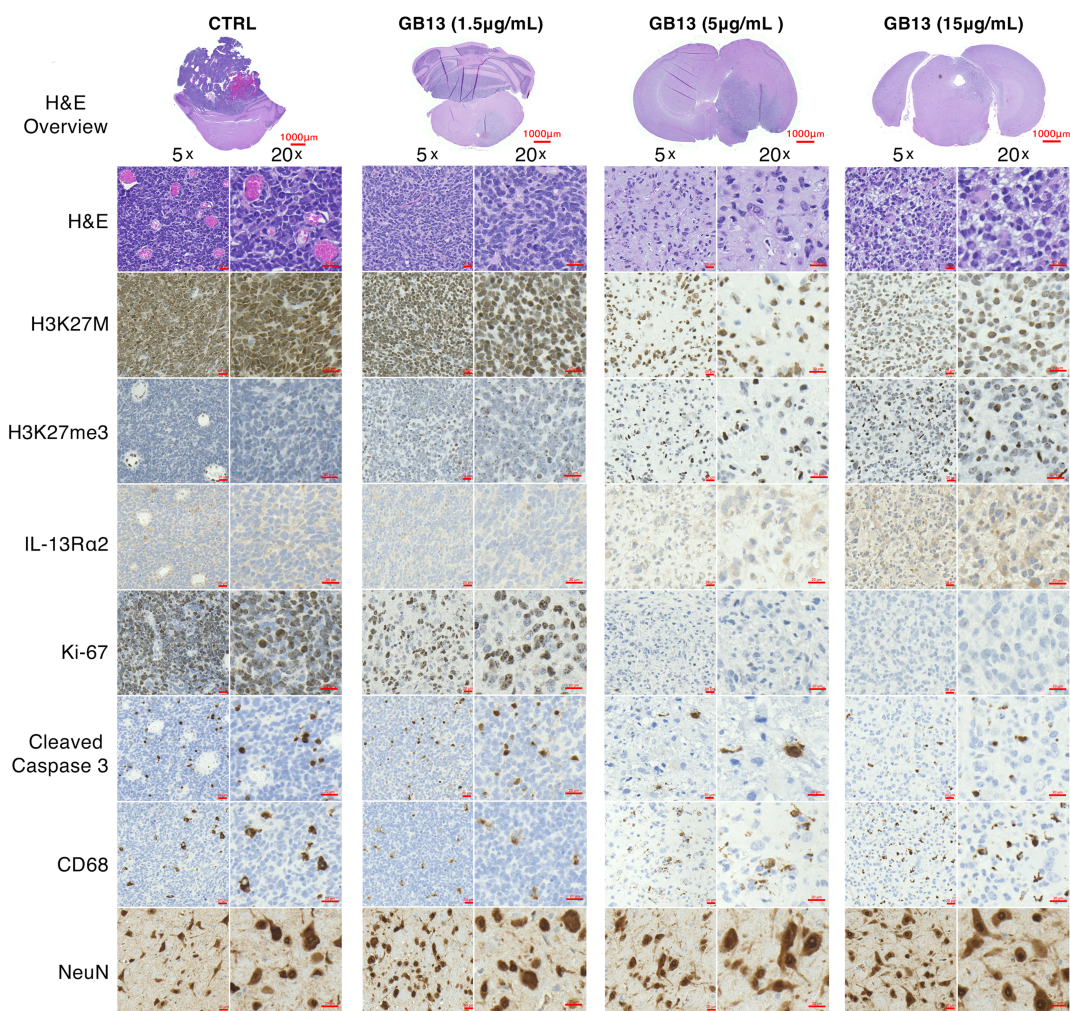
A**B**

Supplementary Figure 5. Convection-enhanced delivery (CED) of GB13 improves the survival of patient-derived orthotopic xenograft (PDOX) DIPG animals. **(A)** Kaplan–Meier graph for mice (n = 3–4 per group) bearing SU-DIPG XVII orthotopic xenografts, treated with 7-day continuous CED of GB13 (1.5 µg/mL, purple curve; 5 µg/mL, wine-red curve; 15 µg/mL, brown curve), compared with vehicle-treated controls (blue curve). $P \leq 0.01$; Log-rank (Mantel-Cox) test. Tx, treatment period. **(B)** Representative Hematoxylin and eosin (H&E) and IHC (H3K27M, H3K27me3, IL-13R α 2, Ki-67, Cleaved Caspase 3, CD68, and NeuN) images of formalin-fixed brain sections from PDOX mice treated with vehicle or GB13 (1.5 µg/mL, 5 µg/mL, or 15 µg/mL). Images are representative of 3–4 mice in each group that were euthanized at the ethical endpoint. Scale bars: 1×: 1000 µm, 5×: 20 µm, 20×: 20 µm.

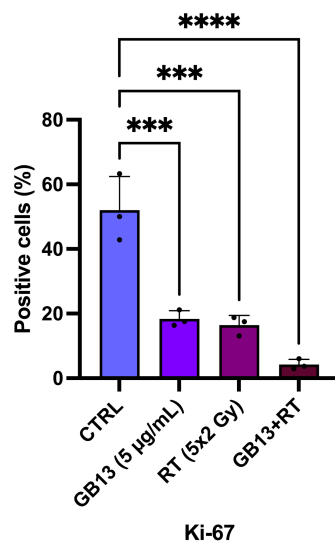
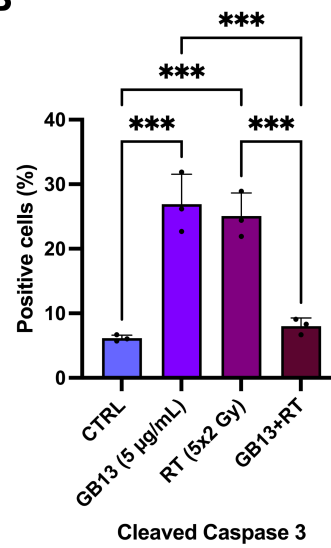
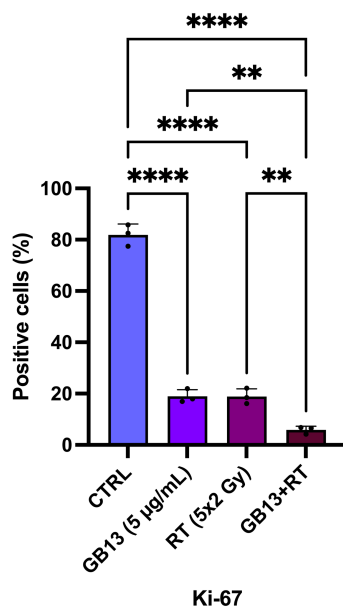
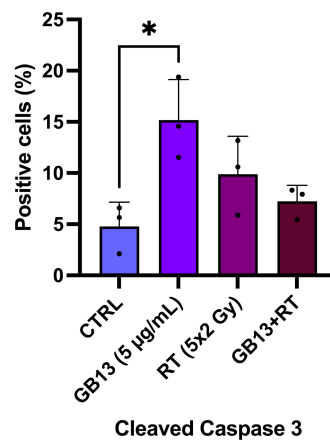
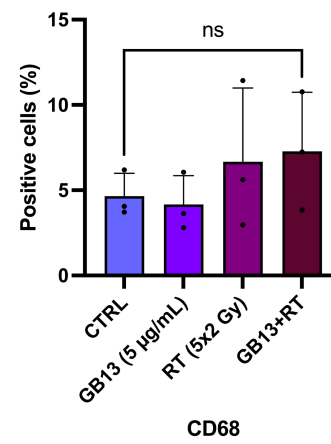
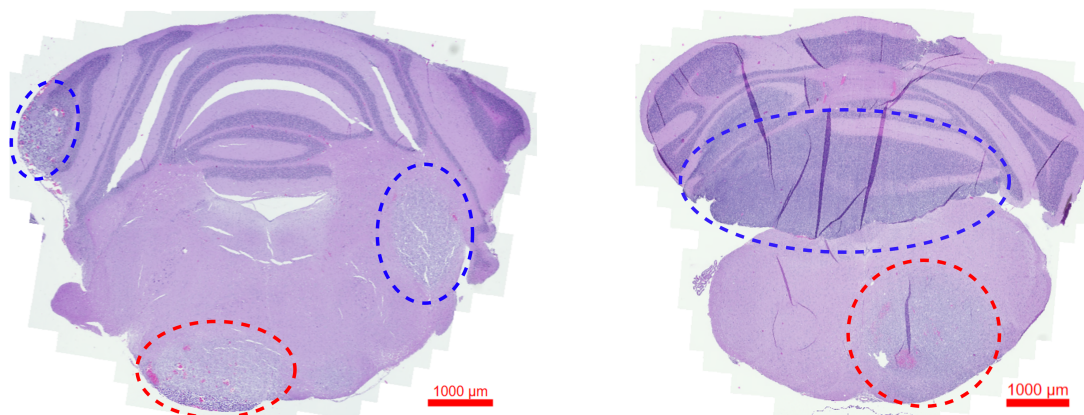
A **H3.3 K27M;PDGFRA;p53^{CKO} GEMM**



B

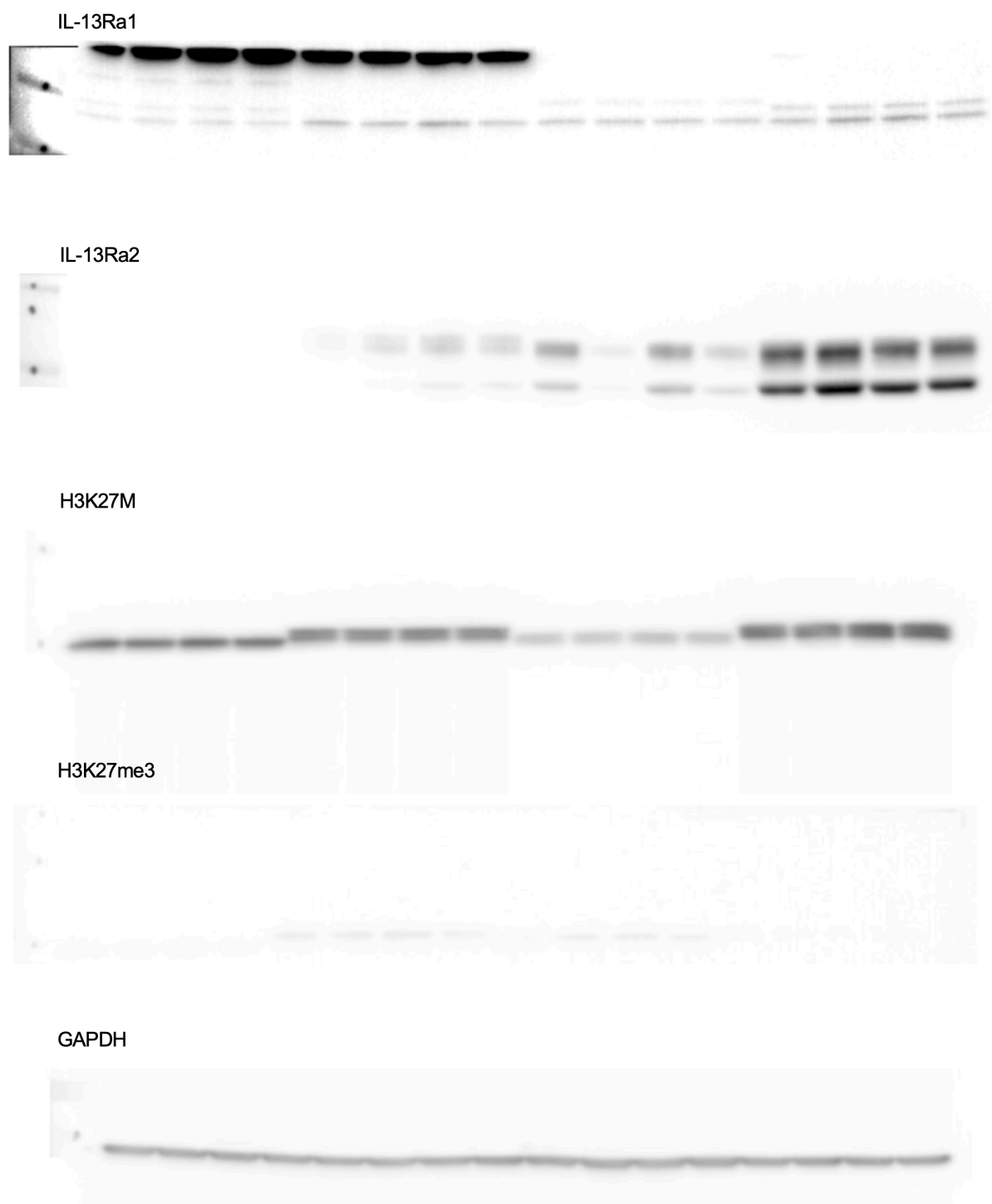


Supplementary Figure 6. CED of GB13 extends the survival of a *H3F3A*-, *TP53*-, and *PDGFRA*-mutant DIPG genetically engineered mouse model (GEMM)⁷. **(A)** Kaplan–Meier graph for mice (n = 4–8 per group) bearing H3.3 K27M;PDGFRA;p53^{cKO} orthotopic tumors, treated with 7-day continuous CED of GB13 (1.5 µg/mL, purple curve; 5 µg/mL, wine-red curve; 15 µg/mL, brown curve), compared with vehicle-treated controls (blue curve). $P \leq 0.01$; Log-rank (Mantel-Cox) test. Tx, treatment period. **(B)** Representative H&E and IHC (H3K27M, H3K27me3, IL-13R α 2, Ki-67, Cleaved Caspase 3, CD68, and NeuN) images of formalin-fixed brain sections from GEMM animals treated with vehicle or GB13 (1.5 µg/mL, 5 µg/mL, or 15 µg/mL). Images are representative of 4–8 mice in each group that were euthanized at the ethical endpoint. Scale bars: 1×: 1000 µm, 5×: 20 µm, 20×: 20 µm.

A**B****C****D****E****F**

Supplementary Figure 7. Concomitant CED of GB13 plus fractionated RT improves the efficacy over either monotherapy in both PDOX and genetically engineered DIPG mouse models. **(A-E)** Immunohistochemical quantification of the time- and treatment-dependent effects of GB13 alone (5 µg/mL), RT alone (2 Gy once per day for 5 consecutive days), or their combination. Treatment-related changes of **(A)** Ki-67 and **(B)** Cleaved Caspase 3 in SU-DIPG XVII orthotopic xenografts. Alterations of **(C)** Ki-67, **(D)** Cleaved Caspase 3, and **(E)** CD68 in mice bearing H3.3 K27M;PDGFRA;p53^{CKO} orthotopic tumors. Results represent the mean ± SD in 4–8 mice per group. ns: $P \geq 0.05$; *: $P \leq 0.05$; **: $P \leq 0.01$; ***: $P \leq 0.001$; ****: $P \leq 0.0001$; one-way ANOVA. **(F)** Representative low-power H&E images of formalin-fixed brain sections from treated GEMM animals. Red dashed lines delineate the perimeter of the tumor area treated by CED, while blue dashed lines indicate tumor regions not targeted by CED. Scale bars: 1000 µm.

Figure 3



Western blot analysis showing protein levels of Mcl-1, Bak, Cleaved Caspase 3, Cleaved PARP, and Total Histone H3 across various cell lines. The blots are arranged vertically, with each row representing a different protein. The lanes are grouped by cell line, with each group containing three replicates. The proteins are labeled on the left side of the image.

C-Myc

Mcl-1

Bak

Cleaved Caspase 3

Cleaved PARP

Total Histone H3

Mcl-1

Bak

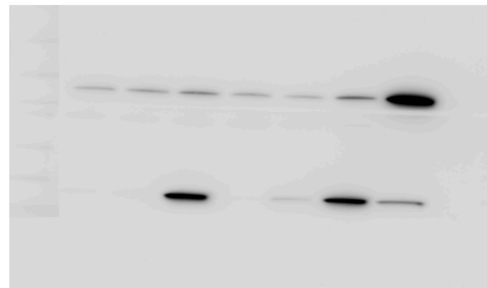
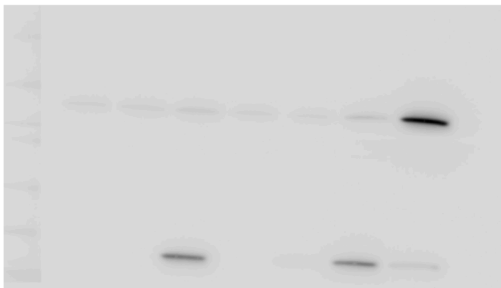
Cleaved Caspase 3

Cleaved PARP

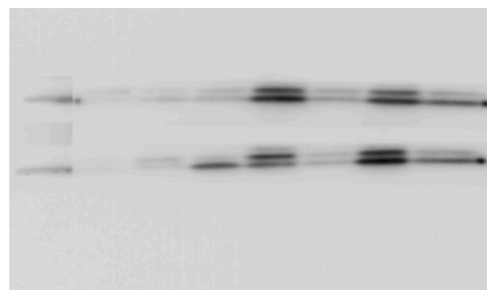
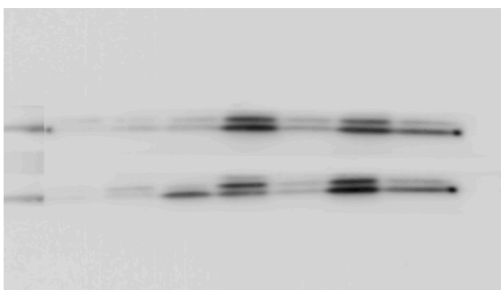
Total Histone H3

Figure 4

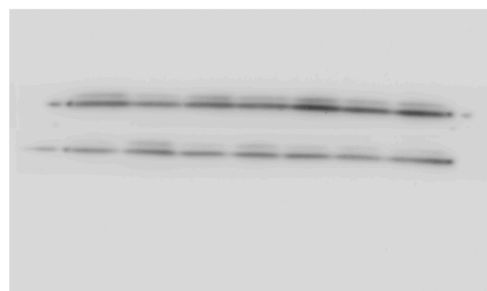
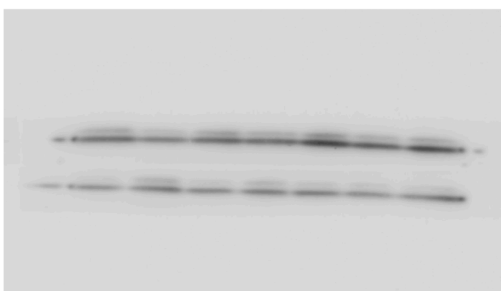
Cleaved PARP



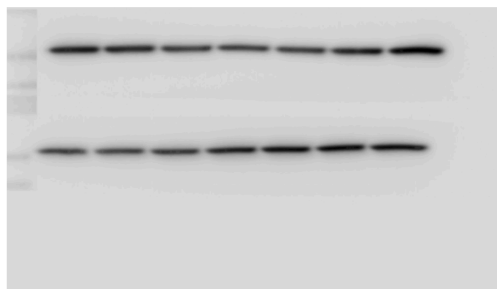
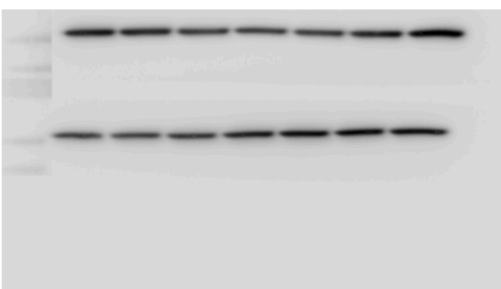
γ H2AX



Total H2AX



α -Tubulin



Supplementary Figure 1

IL-13Ra2



Total Histone H3

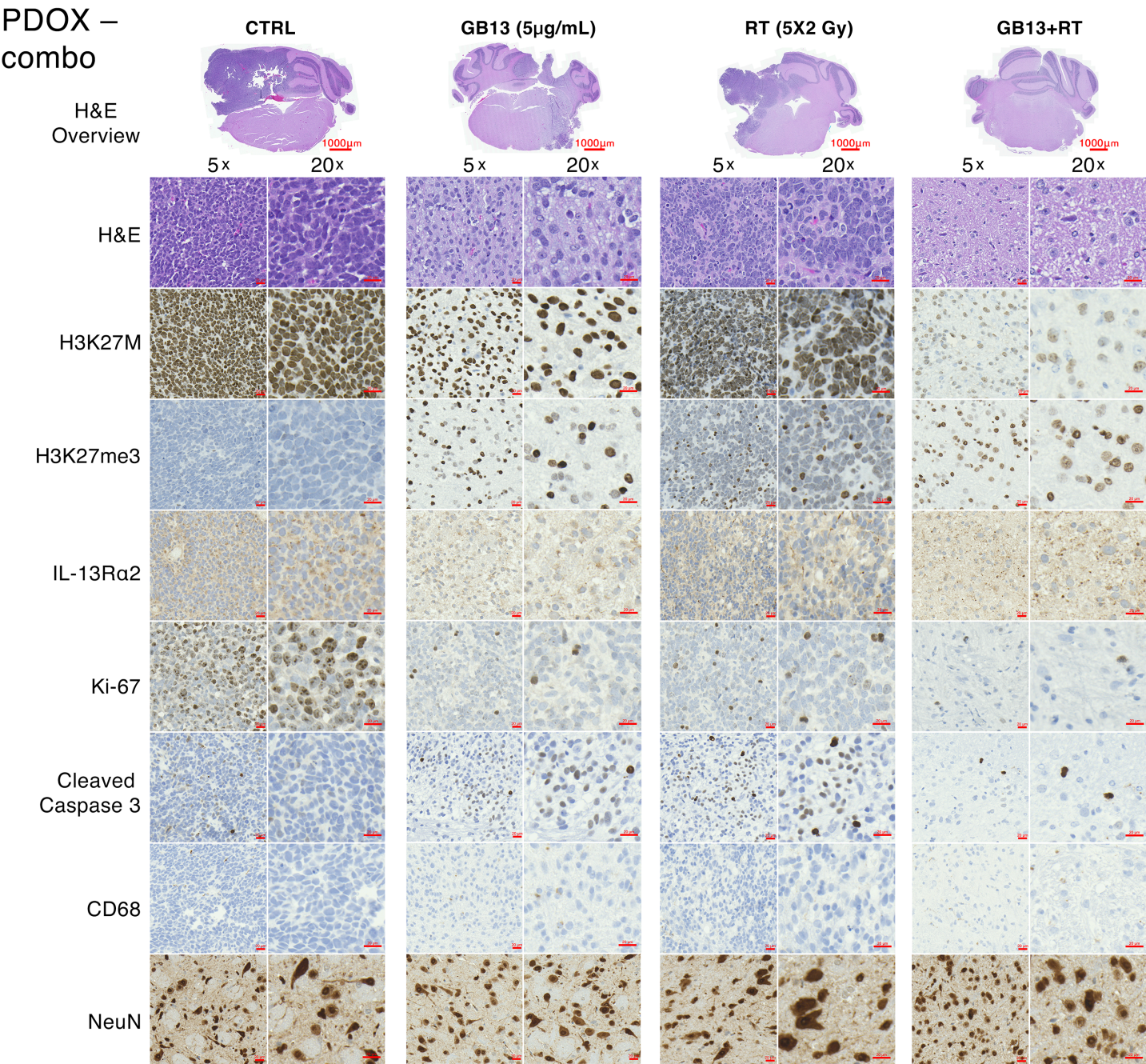


Supplementary Figure 8. Compiled Western blot gels/images indicating treatment modalities, stained antigen, and molecular size of main band as detailed in the Methods and Supplementary Tables section.

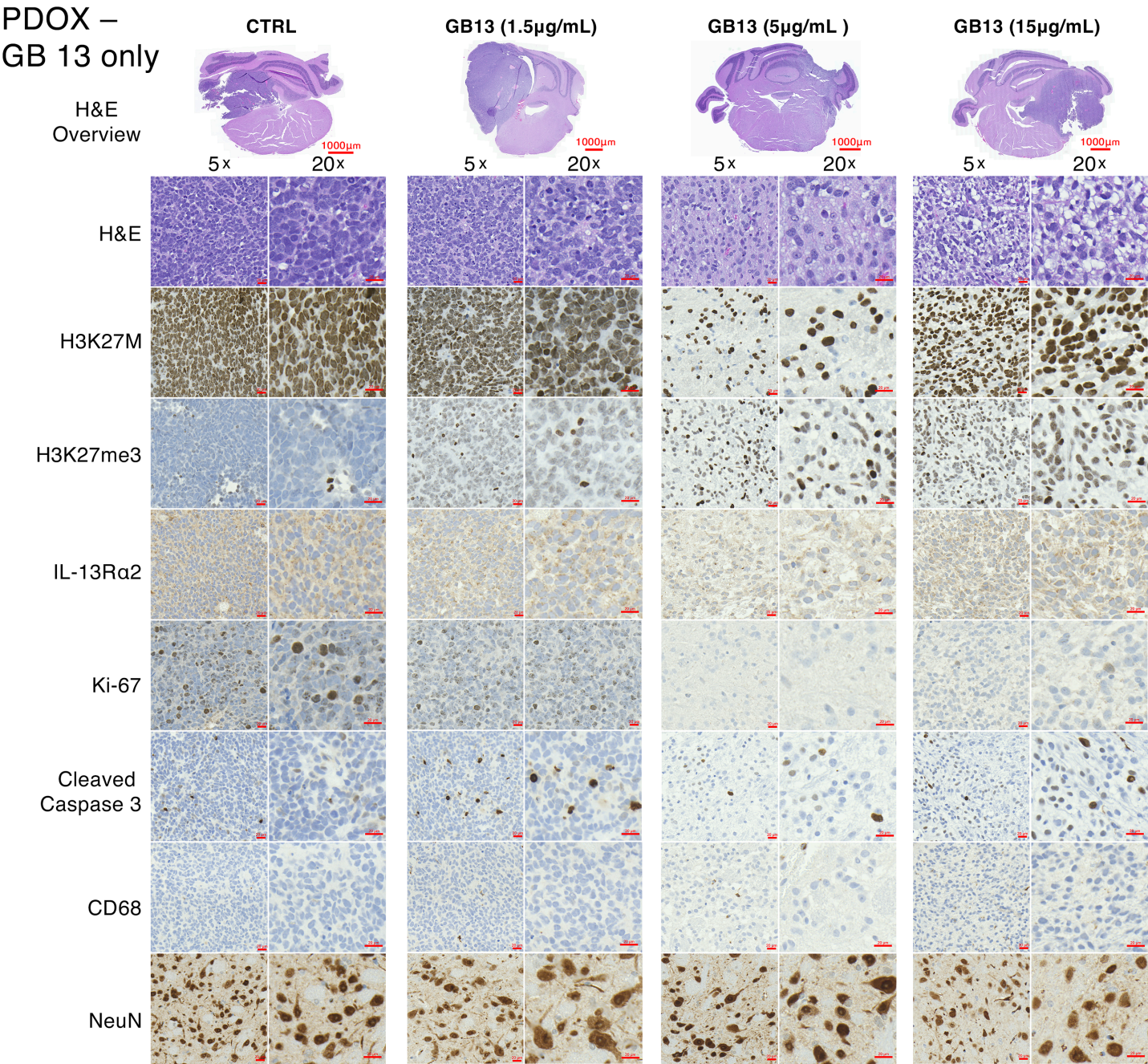
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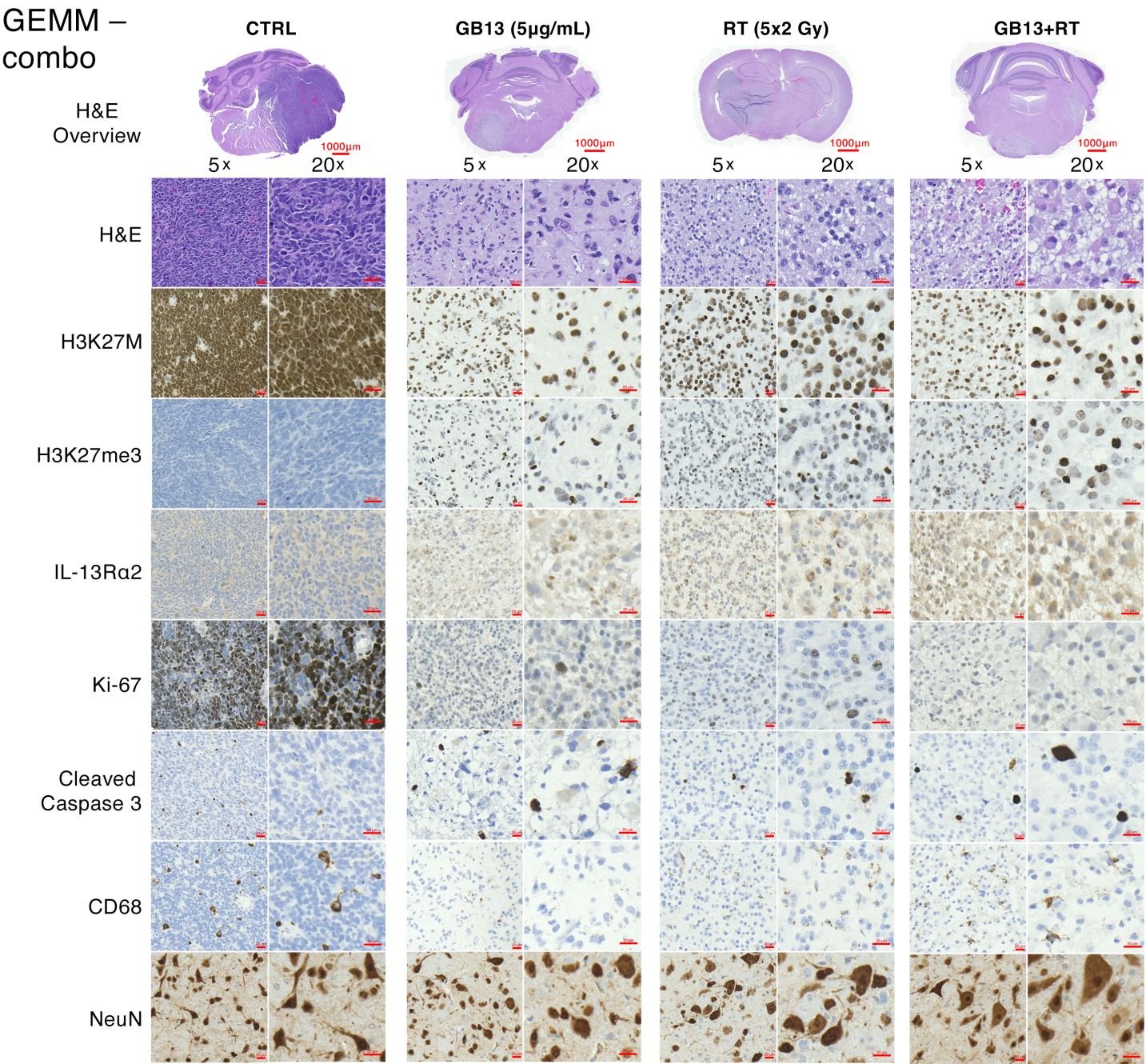
PDOX –
combo



PDOX –
GB 13 only

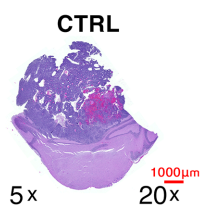


GEMM –
combo

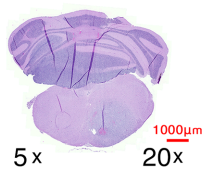


GEMM –
GB 13 only

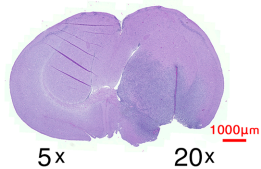
H&E Overview



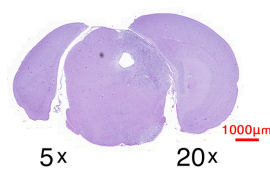
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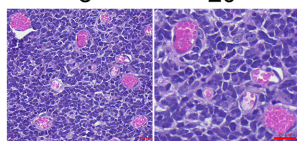
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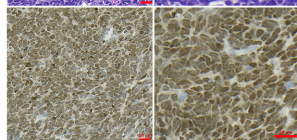
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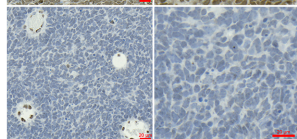
H&E



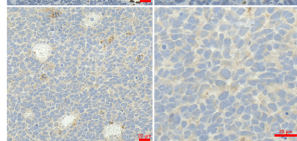
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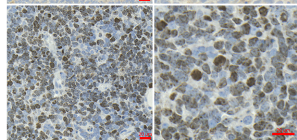
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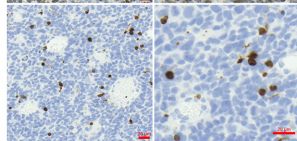
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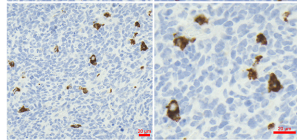
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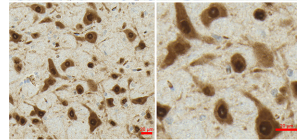
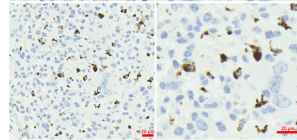
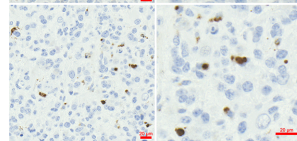
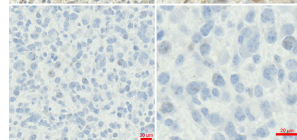
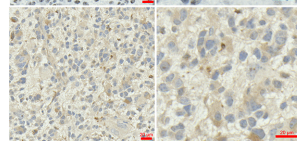
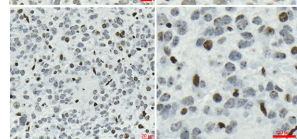
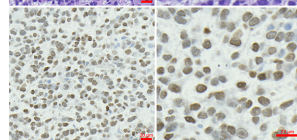
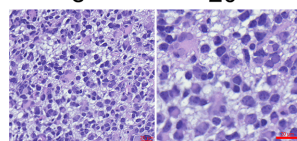
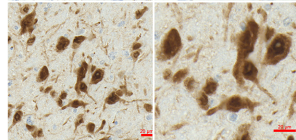
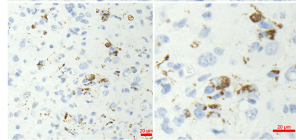
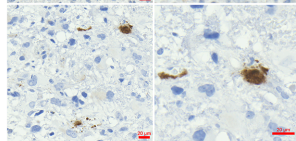
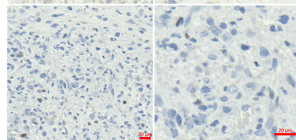
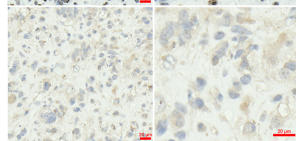
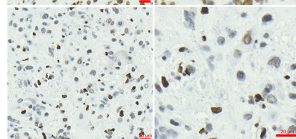
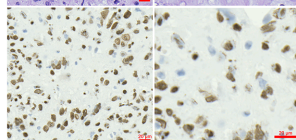
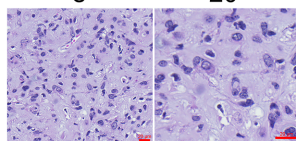
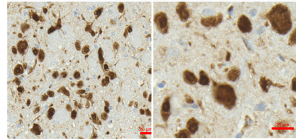
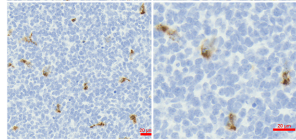
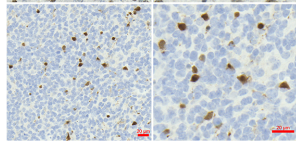
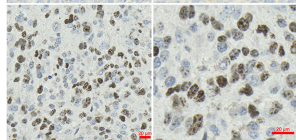
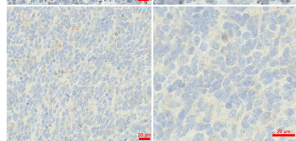
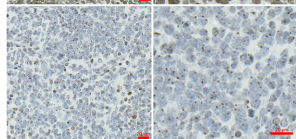
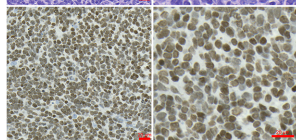
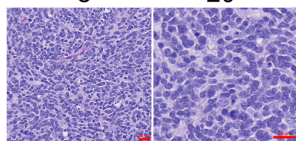
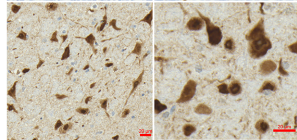
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Caspase 3



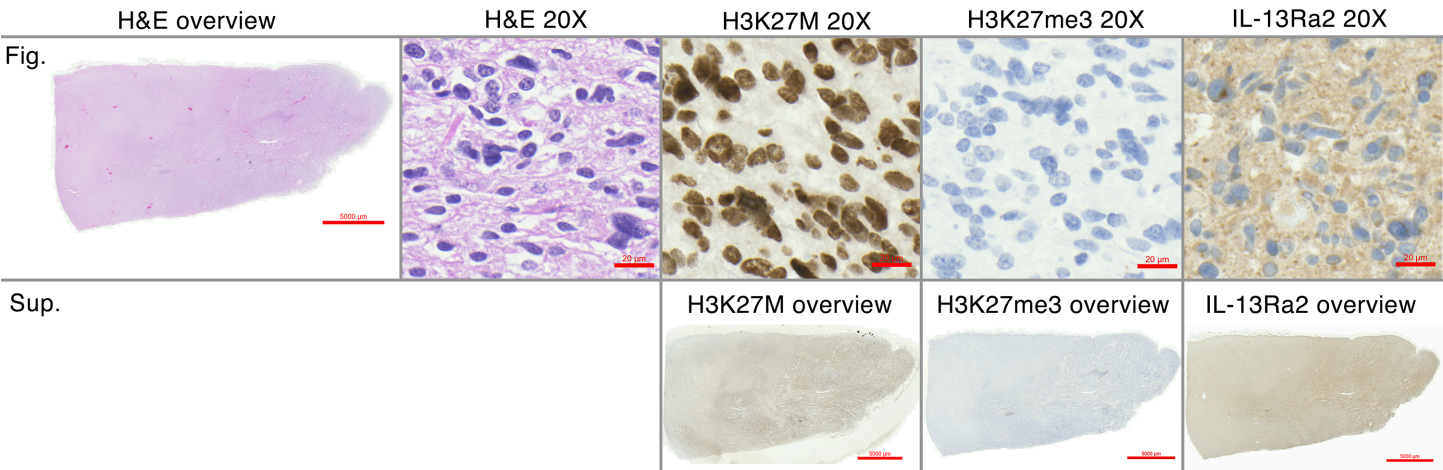
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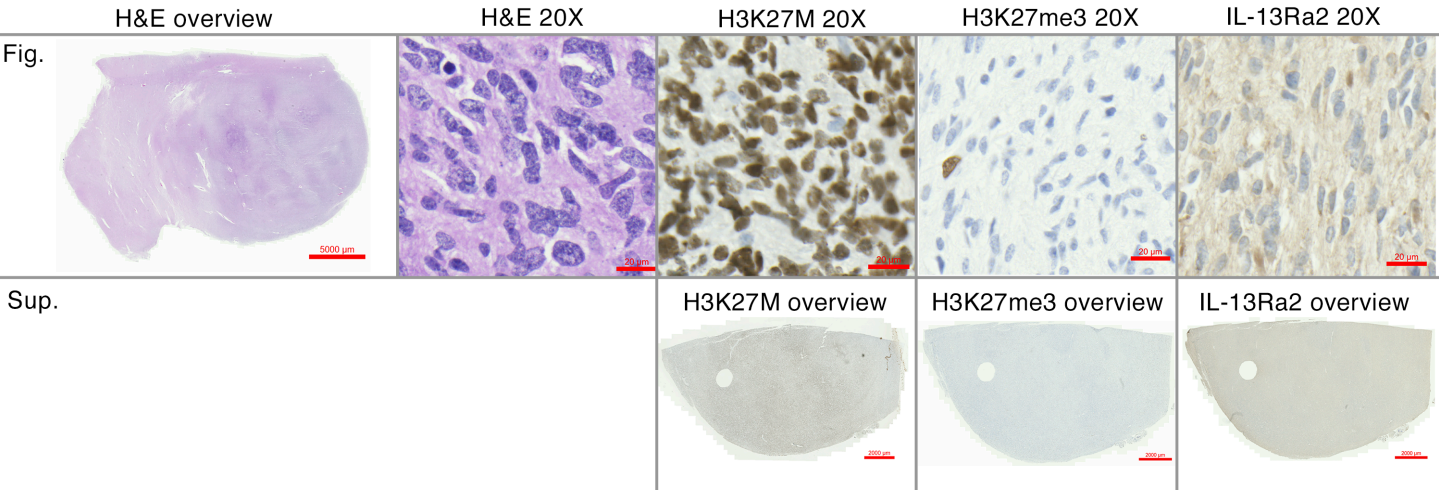
NeuN



NA22-048Q (DIPG+ adjacent normal tissue)



CA22-10 A2 (DIPG+ adjacent normal tissue)



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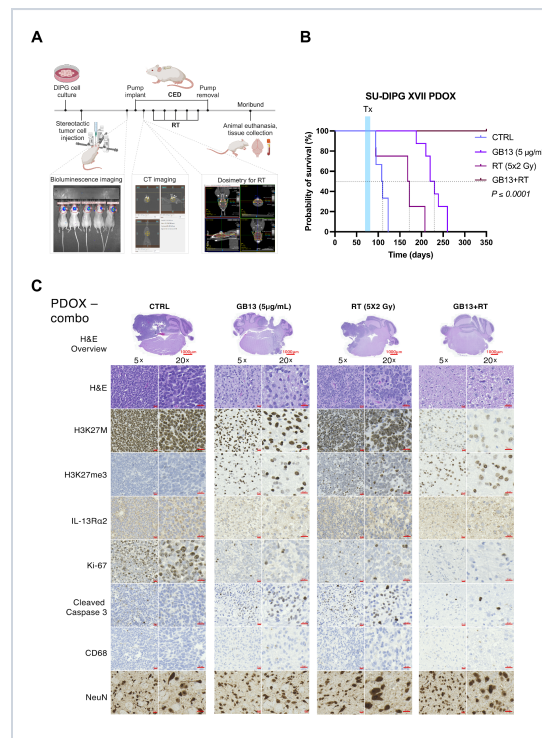
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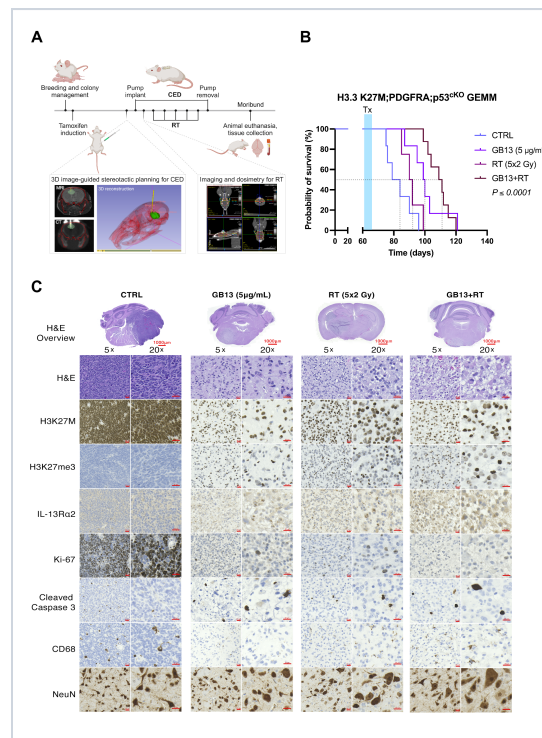
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